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Stability and relapse after orthodontic treatment of deep bite cases—a long-term follow-up study

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SUMMARY The purpose of this long-term follow-up study was twofold—firstly, to assess prevalence of relapse after treatment of deep bite malocclusion and secondly, to identify risk factors that predispose patients with deep bite malocclusion to relapse. Sixty-one former patients with overbite more than 50% incisor overlap before treatment were successfully recalled. Clinical data, morphometrical measurements on plaster casts before treatment, after treatment and at long-term follow-up, as well as cephalometric measurements before and after treatment were collected. The median follow-up period was 11.9 years. Patients were treated by various treatment modalities, and the majority of patients received at least a lower fixed retainer and an upper removable bite plate during retention. Relapse was defined as increase in incisor overlap from below 50% after treatment to equal or more than 50% incisor overlap at long-term follow-up. Ten per cent of the patients showed relapse to equal or larger than 50% incisor overlap, and their amount of overbite increase was low. Among all cases with deep bite at follow-up, gingival contact and palatal impingement were more prevalent in partially corrected noncompliant cases than in relapse cases. In this sample, prevalence and amount of relapse were too low to identify risk factors of relapse.

Introduction

Deep bite, by definition increased overbite, is measured as vertical overlap of the incisors perpendicular to the occlusal plane absolutely in mm, relatively in percentage of incisor overlap or qualitatively by describing the contact of the lower incisors to the upper arch or palate. Most commonly, deep bite can be divided into dento-alveolar origin (overeruption of teeth) and skeletal origin (decreased lower face height, low mandibular plane angle) (Nielsen, 1991).

Deep bite prevalence varies from 8.4 to 51.5%, depending on the threshold values applied, ethnic group and gender (Tausche *et al.*, 2004; Proffit *et al.*, 2007; Lux *et al.*, 2009; Thilander and Myrberg, 1973). Prevalence of palatal non-traumatic tooth contact and palatal impingement was reported to vary from 5.9 to 15.9% (Tausche *et al.*, 2004; Lux *et al.*, 2009). Angle classification (Angle, 1899) has been associated with vertical and/or cephalometric patterns. Class II malocclusion was shown (Lux *et al.*, 2009) to be significantly associated with increased overbite compared with class I malocclusion. Class II Division 2, with a prevalence of 5.3%, a less frequent malocclusion (Ingervall *et al.*, 1978), may be associated with a deep bite (Brezniak *et al.*, 2002). A correlation of increased overbite with retrusive incisors in Angle class I malocclusions as well as in Angle class II Division 2 malocclusions was described in the literature (Simons and Joondeph, 1973).

Treatment of deep bite malocclusion is recommended in order to reduce or prevent tissue trauma from tooth contact (Bjørnaas *et al.*, 1994), facilitate possible future reconstructive dental work and reduce increased tooth wear (Ritchard *et al.*, 1992; Silness *et al.*, 1993). Reoccurrence of malocclusion years after the end of treatment may lead to patients seeking retreatment or questioning the benefit of their initial therapy. Therefore the long-term stability seems to be more important than the final result itself. Relapse is a dento-alveolar and skeletal change after orthodontic treatment towards the initial malocclusion, and it is often encountered even in ideally treated cases (Crum and Andreasen, 1974). These changes are attributed to a physiologic reestablishment of force equilibrium (Proffit *et al.*, 2007), periodontal remodeling (Picton and Moss, 1973; Crum and Andreasen, 1974; Ackerman and Proffit, 1997), growth or normal/abnormal development (Bergersen, 1988; Forsberg *et al.*, 1991; Iseri and Solow, 1996). The loss of about one-third of the orthodontic treatment result during 10 years of follow-up and consistent relapse of all malocclusion characteristics were reported by some authors (Al Yami *et al.*, 1999). Therefore, stability of orthodontic result is one of the biggest challenges in orthodontics.

Increase in overbite after completion of treatment is regarded as relapse for dental deep bite cases. Several authors have described deep bite malocclusions as relapse

prone (Rönnerman and Larsson, 1981; Berg, 1983; Binda *et al.*, 1994). In a study of Berg (Berg, 1983), relapse reduced treatment effect by 18.8% on average in 26 patients with deep bite malocclusion after 5–9 years out of retention. Relapse of deep bite was reported 10 years after treatment in 23 consecutive patients to almost pre-treatment levels, although Hawley plates as retention devices were used in most of the patients (Rönnerman and Larsson, 1981). Lapatki *et al.* (2004) investigated a sample with deep bite and retroclined incisors and found 20% of vertical relapse on median 2 years post-treatment. Mean relapse below 1 mm is reported by two investigations 7 or 8.2 years after treatment (Canut and Arias, 1999; Schütz-Fransson *et al.*, 2006; Al-Buraiki *et al.*, 2005). Several findings regarding deep bite relapse are reported from studies investigating samples with other malocclusions. The curve of Spee in a class II Division 1 sample was reported to gradually relapse over the years or decades in 21 out of 31 cases (Bernstein *et al.*, 2007). Similarly, a positive correlation between years out of retention and overbite relapse was found in another class II Division 2 sample (Canut and Arias, 1999). In 80% of short facial type patients a post-treatment increase in overbite was recorded 2 years after treatment completion despite the use of removable retention for 1 year (Zaher *et al.*, 1994).

Factors that play a role in the development of deep bite may also play a role in the development of relapse, such as growth (Björk, 1969; Simons and Joondeph, 1973; Driscoll-Gilliland *et al.*, 2001; Baccetti *et al.*, 2011), function (Lapatki *et al.*, 2007; Sciote *et al.*, 2012), incisor overeruption (Burstone, 1977; Lowe *et al.*, 1986) or hypodontia (Dermaut *et al.*, 1986). Only a few analyses of which factors lead to more stable results or which are associated with relapse exist. Preston *et al.* (2008) did not find a significant correlation between pre-treatment severity of malocclusion and relapse and no ability to predict relapse by mandibular intercanine width, overbite, overjet, mandibular incisor irregularity and arch length. Regardless of the treatment modality the authors found a significant higher prevalence of relapse for patients in whom the dentitions were not completely leveled at the end of the treatment. Several authors (Millett *et al.*, 2006; Preston *et al.*, 2008) did not show differences in relapse regarding the type of treatment. One study correlated protrusion of mandibular incisors during orthodontic treatment with overbite relapse (Simons and Joondeph, 1973). Neither a systematic review about retention of deep bite or stability/relapse of deep bite does exist nor it is possible today to predict risk of relapse for an individual after therapy of deep bite.

Therefore the primary purpose of this long-term follow-up retrospective study was to assess the prevalence of deep bite relapse in a sample of former orthodontic patients. The secondary purpose was to identify important factors in relapse of deep bite to develop hypotheses for a future prospective clinical trial.

Material & Methods

The sample of the present retrospective study consisted of patients treated at the Department of Orthodontics and Dentofacial Orthopedics, University of Bern, Switzerland. No standardized treatment or retention protocols were used at that time, but an individual treatment and retention plan was established for each case by different postgraduate students and supervisors. Two investigators (D and B) selected the records, which were stored in the archive during the years 2000 to 2002. The inclusion criteria were overbite of 50% or greater as measured as overlap of the incisors on the initial study models with pencil and ruler (Nanda, 1981).

Of a total of 855 former patients, 185 (22%) who met the inclusion criteria were contacted through mail or electronic search of the official phone book by name, address or phone number of the patient himself/herself or the parents by two investigators (A and B). Due to the long follow-up period, patients were found to have moved, and contact was lost with 98 former patients, of whom two had died. With the agreement of the independent local research ethics committee Bern, Switzerland (KEK Nr. 036/10, 27.04.2010), we were able to contact personally 87 patients. Sixty-one (70%) were willing to participate, whereas 26 (30%) refused to participate for various reasons. For each of the consenting patients, tooth cleaning was provided, their retention appliances were examined, and new impressions for dental casts were taken by three investigators (B, A, D). From the 61 participants, 18 had to be excluded due to the following reasons: 3 had missing models at T1 and/or T2, 7 had OB < 50% at T1, 1 had retreatment during the follow-up period, 7 had partial treatment (T2 and T3 OB > 50%). These seven patients who discontinued treatment were assigned to the partial treatment group. The assignment process as well as drop outs are depicted in a flow chart in Figure 1.

The patient records consisted of three sets of dental casts: pre-treatment (T1), post-treatment (T2), and end of follow-up (T3). All complete sets of two lateral cephalograms at T1 and T2 were evaluated.

Measurements of lateral cephalometric radiographs

One investigator (C) evaluated the T1 and T2 cephalometric radiographs of the patients using cephalometric software (Viewbox 4, dHAL Software, Kifissia, Greece). The radiographs were scanned at 300ppi, and the following angles were measured: SNA, SNB, ANB, SN-GoGn, PP-MP, Gonial angle, U1-PP, U1-APog, L1-MP, L1-APog and Interincisal angle. Additionally, the following ratios were calculated: Lower Face Height / Total Face Height, Posterior Face Height / Anterior Face Height (S-Go/N-Me).

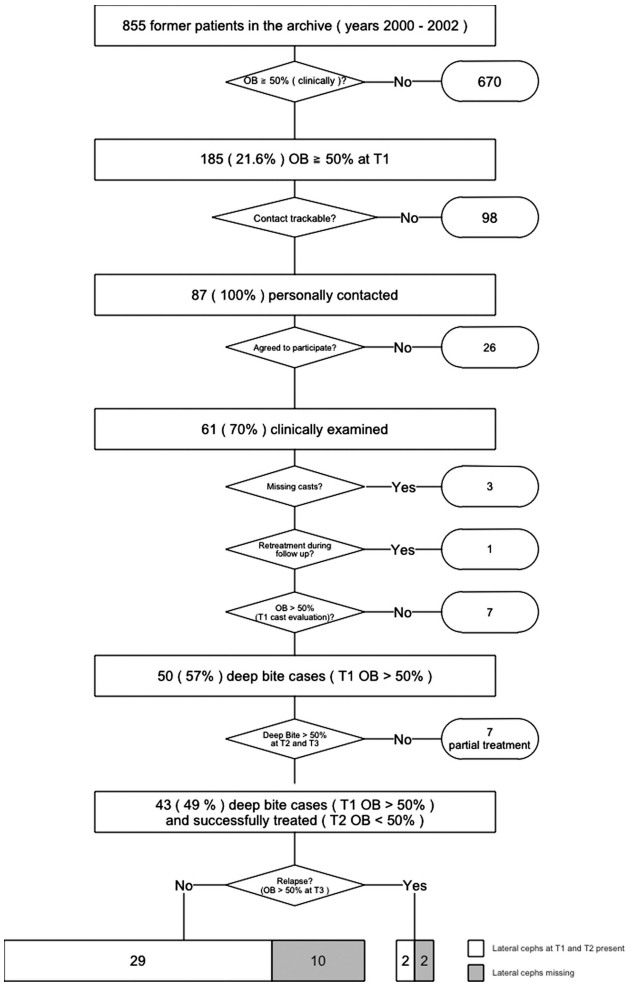

Figure 1 Flow chart of patient selection according to inclusion and exclusion criteria. Inclusion criteria OB > 50% at T1 was applied twice. The first time with pencil and ruler in the archive and a second time with a digital sliding caliper. Finally the remaining deep bite cases were assigned to the relapse group and the stable group.

Measurements on dental casts

Measurements on T1-T3 plaster models were carried out by one investigator (C), using a fine tip digital caliper (150 mm ISO 9001 electronic caliper, Tesa Technology, Renens, Switzerland). The following measurements were made: maxillary and mandibular intercanine width, maxillary and mandibular intermolar width between the central fossae, incisor overjet, upper incisor overlap (percentage of the lower incisor overlap by the upper incisor, measured perpendicular to the occlusal plane).

Statistical Analysis

Descriptive statistics were performed at T1, T2, and T3. Medians were preferred to the means because of the higher robustness against outliers with small sample numbers.

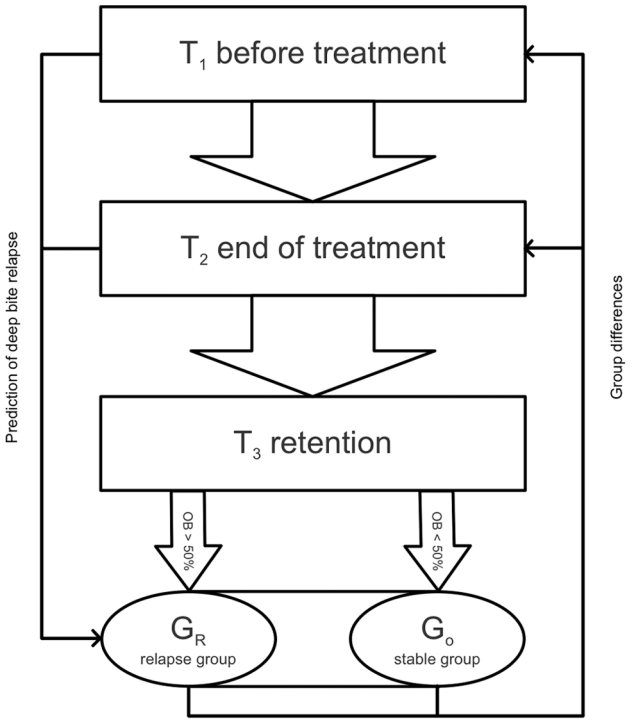

Figure 2 The study was planned with the intention to predict relapse by clinical, cast related or cephalometric variables at T1 or T2 by logistic regression analysis. Amount and risk of relapse was too low for inferential statistics.

The initial aim was to divide the sample into a relapse and a non-relapse group and to identify risk factors predicting the relapse of deep bite using a logistic regression model as shown in Figure 2. The threshold value was defined at initiation of the study according to the inclusion criteria as 50% upper incisor overlap. The outcome variable 'upper incisor overlap at T3' would have been used to test the hypothesis that 'there is no difference between the relapse and the non-relapse group'. Given the small number of patients in the relapse group, a comparison of the groups or a logistic regression analysis was not feasible. Therefore, only descriptive analysis is presented in this article.

Error of the method

The intracluster correlation coefficient (ICC) was used in order to assess the method error and specifically intra-examiner agreement. The ICC was calculated for all the variables measured on 20 randomly selected dental casts and 20 randomly selected cephalometric radiographs using the Stata 12.1 statistical package (Stat Corp, College Station, TX, USA).

Results

The ICC ranged for both dental casts and measurements ranged from 0.94 to 0.99, indicating very low measurement error/excellent intra-rater agreement.

The median age was 12.5 and 11.5 years at T1, 17.1 and 16.7 years at T2, and 29.2 and 26.9 years at T3 for the relapse and the non-relapse group, respectively. The median active treatment duration was 3.2 years in the relapse group and 3.6 years in the non-relapse group, while the median long-term follow-up period was 13.4 years (range 10.7 years to 16.5 years) and 11.7 (range 9.5 to 16.4 years), respectively. The median long-term follow-up period of all cases was 11.9 years (range 9.5 years to 16.5 years). Clinical data, cast analysis, and cephalometric evaluation are summarized in Table 1.

Four out of 43 cases (10.3%) showed relapse of the incisor overlap of equal to or larger than 50.0% during the long-term follow-up period. An example of each group is shown in Figures 3 and 4. The relapse group consisted of three females and one male, three cases with extractions or missing teeth, and one case with all teeth present; none of the patients had an upper fixed retainer at T3, and two patients had a lower fixed retainer at T3. Sixty-five per cent of patients had a removable retention appliance after active treatment for a median period of 1.4 years (range 0.2 to 4.4 years); in the relapse group, one patient received a removable retention appliance. The incisor overlap at T3 in the relapse group ($N = 4$) ranged from 55.6 (4.8 mm) to 50.0% (3.9 mm) and in the non-relapse group ($N = 39$) from 48.5 (4.7 mm) to 0.0% (0.0 mm). During the long-term follow-up period the incisor overlap increased in the relapse group by 6.7% (median, range 3.2% to 19.8%) and decreased on average in the non-relapse group by −1.3% (median, range from 13.8 to −26.3%). The change of the overjet during the long-term follow-up period was for the relapse group −1.8 mm (median, range 0.3 to −2.8 mm) and for the non-relapse group 0.1 mm (median, range from −1.4mm to 2.0 mm).

The partial treatment group ($N = 7$) had significantly increased prevalence of gingival contact or palatal impingement at T3 compared with the complete treatment group (relapse and non-relapse pooled, $N = 50$, Fishers exact test $P < 0.01$) as shown in Table 2. Two examples are shown in Figures 5 and 6.

Discussion

The prevalence of vertical relapse 12 years after orthodontic treatment of moderate deep bite was found to be low in the present study. Relapse was defined as an increase in incisor overlap >50% during follow-up. 10% of the patients showed relapse with a low median increase of 6.7%, while 90% showed normal vertical relations at long-term follow-up.

Our findings are in agreement with those of another study, carried out on a sample with similar inclusion criteria, treatment and retention protocols (Schütz-Fransson *et al.*, 2006). In the cases with moderate dento-alveolar deep bite and successful treatment with subsequent retention by fixed retainers and a temporary removable upper plate, the prevalence and degree of deep bite relapse were relatively small and clinically insignificant.

The low prevalence and amount of relapse may be attributed to the relatively high median age at T2 (17 years) of our sample (Iseri and Solow, 1996). While it is not scientifically proven by longitudinal superimpositions, it is plausible that more remaining growth in addition with anterior growth rotation (Björk and Skieller, 1983) and without retention would experience increased relapse. A recent study compared facial types and found a lower relapse tendency for high angle patients compared with normal or low angle patients (Pollard *et al.*, 2012). The treatment duration was relatively long, indicating that most of the periodontal remodeling (Crum and Andreasen, 1974; Kilic *et al.*, 2011) had already taken place at the time of debonding, and it is not expected that it would have contributed substantially to the relapse.

Treatment with removable appliances (Hans *et al.*, 1994), fixed appliances with or without extractions (Parker *et al.*, 1995) and in severe cases also with maxillofacial surgery to correct deep bite have been proposed. Various treatment modalities and combinations were used in this sample, including one case with maxillofacial surgery (Figure 7). There are three theoretical ways to orthodontically treat deep bite malocclusion by leveling of the arch/curve of Spee: (1) intrusion of lower and/or upper incisors (Burstone, 1977; Nanda, 1981; Ng *et al.*, 2005), (2) labial inclination of the incisors (pseudo-intrusion) (Burstone, 1977), and (3) extrusion of posterior teeth possibly associated with a clockwise rotation of the mandible, which would increase lower face height (Nanda, 1981). This theoretical clockwise rotation does not seem to occur in all cases (Bernstein *et al.*, 2007). No evidence-based recommendations regarding the effectiveness of treatment of class II Division 2 malocclusion in children can be deducted from the existing literature (Millett *et al.*, 2006). Stability of deep bite was achieved by various treatment modalities and combinations.

Three subjects of the relapse group showed spacing in the upper front (two central diastemae), and the fourth had increased overjet due to a unilateral distal occlusion. All the four relapse subjects were missing upper fixed retainers at T3. It could be that development of spacing in the upper jaw or persistent overjet with missing dental incisor contact allowed deepening of the bite in these relapse cases. The influence of lower lip pressure and height of the lip line was not investigated in this study. Secondary malocclusion (Williams *et al.*, 1982; Artun and Urbye, 1988; Lindhe *et al.*, 2003) due to pathologic tooth migration related to periodontal disease or pronounced attachment loss is unlikely at the age of this sample but may become more relevant later in life.

With regard to the four relapse cases and the partial treatment group, we can speculate that absence of fixed retainers, relapse of arch form (loss of arch length and crowding of lower incisors) (Stenvik *et al.*, 2011) or development of upper spacing may facilitate deepening of the bite. Without retention, other co-factors like the initial severity

Table 1 Descriptive analysis for clinical, dental cast and cephalometric measurements stratified by stable and relapse group.

	Stable			Relapse		
Clinical data	*n*	Median	Range	*n*	Median	Range
Age T1 (years)	39	11.5	9.0–40.8	4	12.5	10.2–14.0
Age T2 (years)	39	16.7	13.2–45.1	4	17.1	15.4–21.2
Age T3 (years)	39	26.9	23.3–56.4	4	29.2	26.4–30.3
Active treatment (years)	39	3.6	0.9–7.9	4	3.2	1.6–4.0
Long-term follow-up (years)	39	11.7	9.5–16.4	4	13.4	10.7–16.5
	n	% (of 39)		*n*	% (of 4)	
Extracted or missing teeth	18	46		3	75	
Upper removeable plate	27	69		1	25	
Upper fixed retainer at T3	14	36		0	0	
Lower fixed retainer at T3	31	79		2	50	
Cast analysis	*n*	Median	Range	*n*	Median	Range
Overjet T1 (mm)	39	5.7	0.6–11.8	4	4.2	0.9–12.2
Overjet T2 (mm)	39	2.6	1.3–4.8	4	3.9	2.3–5.7
Overjet T3 (mm)	39	2.3	1.2–4.6	4	2.5	1.6–3.2
Overjet T2-T1 (mm)	39	−2.8	−9.7–1.0	4	−1.7	−7.0–4.8
Overjet T3-T2 (mm)	39	0.1	−1.4–2.0	4	−1.8	−2.8–0.3
Incisor overlap T1 (%)	39	64.2	50.7–115.6	4	60.2	50.9–75.1
Incisor overlap T2 (%)	39	28.4	13.6–60.7	4	47.3	30.7–49.6
Incisor overlap T3 (%)	39	28.8	0.0–48.5	4	52.9	50.0–55.6
Incisor overlap T2-T1 (%)	39	−33.7	−89.6–3.2	4	−20.0	−30.2–−1.3
Incisor overlap T3-T2 (%)	39	−1.3	−26.3–13.8	4	6.7	3.2–19.8
T1 mand. ICD (mm)	21	25.5	19.9–28.8	3	26.5	24.2–27.1
T2 mand. ICD (mm)	39	26.8	22.1–29.9	4	25.0	24.2–27.1
T3 mand. ICD (mm)	39	26.5	21.3–29.1	4	24.4	23.6–27.6
T1 max. ICD (mm)	16	32.6	29.4–39.3	1	30.7	30.7–30.7
T2 max. ICD (mm)	39	35.1	29.9–38.4	4	31.3	25.2–36
T3 max. ICD (mm)	39	34.8	29.8–38.4	4	31.0	24.5–35.7
T2-T1 max. ICD (mm)	16	1.5	−5.8–4.9	1	−5.5	−5.5–−5.5
T2-T1 mand. ICD (mm)	21	0.6	−2.7–4.8	3	−1.7	−2.4–0.5
T3-T2 max. ICD (mm)	39	−0.1	−1.3–2.0	4	−0.4	−0.6–−0.2
T3-T2 mand. ICD (mm)	39	−0.2	−5.5–2.1	4	−0.5	−0.9–0.5
Lateral ceph analysis	*n*	Median	Range	*n*	Median	Range
SNA T1 (°)	29	79.5	73.0–89.1	2	78.5	77.7–79.2
SNA T2 (°)	29	77.5	69.6–88.0	2	76.4	75.2–77.5
SNA T2-T1 (°)	29	−1.4	−4.9–1.7	2	−2.1	−2.5–−1.7
SNB T1 (°)	29	75.4	68.7–82.9	2	75.5	75.2–75.7
SNB T2 (°)	29	75.7	69.7–85.7	2	75.1	74.7–75.6
SNB T2-T1 (°)	29	0.7	−3.2–3.9	2	−0.3	−0.5–−0.1
ANB T1 (°)	29	4.2	−1.5–8.3	2	3.0	2.4–3.5
ANB T2 (°)	29	1.8	−2.6–6.6	2	1.2	0.5–1.9
ANB T2-T1 (°)	29	−1.8	−6.5–0.8	2	−1.8	−1.9–−1.6
GoGn-SN T1 (°)	29	33.7	21.1–44.7	2	32.5	30.6–34.4
GoGn-SN T2 (°)	29	33.0	17.0–44.5	2	33.3	28.4–38.2
GoGn-SN T2-T1 (°)	29	−1.1	−4.1–4.9	2	0.8	−2.2–3.8
MP T1 (°)	29	23.6	9.5–32.5	2	23.0	16.2–29.8
MP T2 (°)	29	22.4	11.0–34.3	2	22.9	14.2–31.5
MP T2-T1 (°)	29	−1.1	−4.8–6.7	2	−0.2	−2.0–1.7
LFH / TFH T1 (%)	29	54.0	50.1–59.3	2	54.7	54.4–55.0
LFH / TFH T2 (%)	29	55.6	52.9–60.6	2	55.2	54.2–56.2
LFH / TFH T2-T1 (%)	29	0.9	−0.8–3.2	2	0.5	−0.2–1.2
PFH / AFH T1 (%)	29	60.3	52.1–73.2	2	63.5	63.3–63.6
PFH / AFH T2 (%)	29	62.4	56.3–79.7	2	63.5	61.4–65.6
PFH / AFH T2-T1 (%)	29	1.7	−5.8–6.9	2	0.1	−1.9–2.0
U1-PP T1 (°)	29	106.9	79.1–127.2	2	98.9	95.5–102.2
U1-PP T2 (°)	29	109.9	100.5–127.7	2	108.6	103.0–114.1
U1-PP T2–T1 (°)	29	3.2	−17.3–38.1	2	9.7	7.5–11.9
L1-MP T1 (°)	29	86.9	78.7–101.2	2	83.3	82.4–84.1
L1-MP T2 (°)	29	92.8	83.4–107.6	2	85.0	83.2–86.8
L1-MP T2-T1 (°)	29	5.7	−8.7–13.7	2	1.8	0.8–2.7
Interincisal angle T1 (°)	29	136.7	109.1–182.3	2	147.9	144.9–150.8

(*Continued*)

Table 1 (Continued)

	Stable			Relapse		
Clinical data	*n*	Median	Range	*n*	Median	Range
Interincisal angle T2 (°)	29	130.6	102.7–144.1	2	136.5	136.3–136.7
Interincisal angle T2-T1 (°)	29	−7.2	−55.2–12.8	2	−11.4	−14.1–−8.6
Gonial angle T1 (°)	29	122.9	110.0–140.8	2	124.3	121.2–127.4
Gonial angle T2 (°)	29	119.6	109.0–134.8	2	123.9	120.0–127.7
Gonial angle T2-T1 (°)	29	−1.8	−10.9–12.8	2	−0.5	−1.2–0.3
L1-APog T2 (mm)	29	1.5	−2.0–7.8	2	−0.2	−1.8–1.4
L1-APog T1 (mm)	29	−0.6	−9.1–2.7	2	−1.9	−2.9–−0.8
L1-APog T2-T1 (mm)	29	2.6	−0.7–10.5	2	1.7	1.1–2.2
U1-APog T2 (mm)	29	4.4	1.9–10.2	2	2.8	2.0–3.5
U1-APog T1 (mm)	29	5.5	−4.6–10.8	2	1.1	−0.9–3.0
U1-APog T2-T1 (mm)	29	−0.8	−6.0–9.0	2	1.7	0.5–2.9

Differences in numbers (N) are due to missing permanent canines or missing lateral cephalograms.

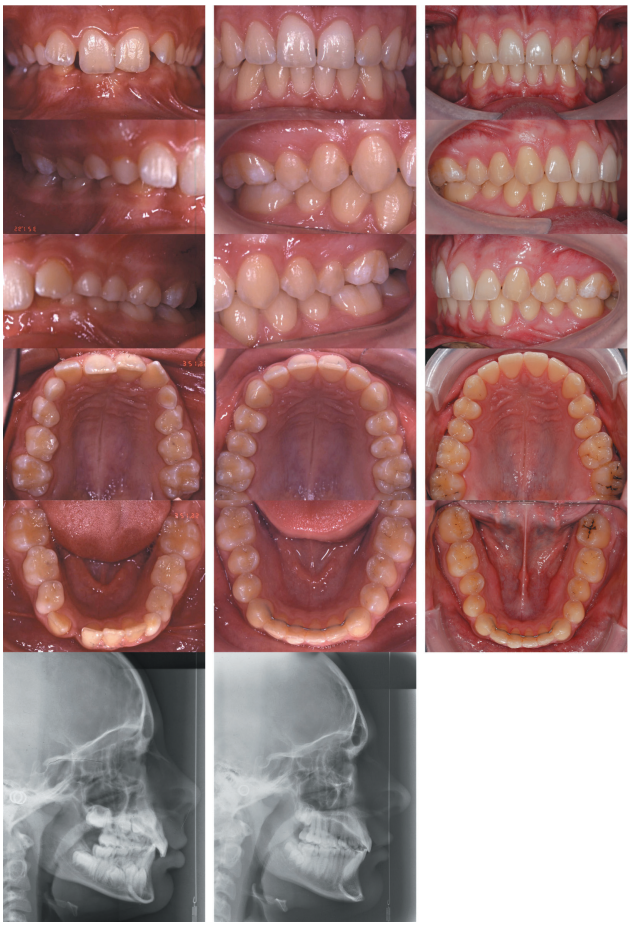

Figure 3 Long-term stable case. Angle class II Division 2, skeletal class II, deep bite with palatal impingement, skeletal hypodivergency, moderate space deficency in the lower arch (left column). Correction with functional appliance and headgear, Goshgarian, multibracket appliance segmented arch technique with base arch, upper retention plate, dental incisor contact (middle column). 10 years long term follow-up (right column).

Figure 4 Relapse case with biggest incisor overlap at T3. Angle class II Division 1, skeletal class II, deep bite with gingival incisor contact, skeletal hypodivergency, spacing in the upper front (left column). Treatment with a removable plate with a frontal bite plateau and headgear. The patient rejected fixed treatment despite slightly increased overjet, spacing in the upper front and missing incisor contact. No fixed retainers were used after treatment (middle column). 10 years later relapse of incisor overlap is apparent but now seemingly stable with frontal dental contact and no complaints.

Table 2 Type of lower incisor contact at T3 in the complete treatment group (relapse and non-relapse group pooled) and the partial treatment noncompliance group (excluded cases due to OB > 50% at T2 and T3) in which overbite has never been treated successfully.

	Complete Treatment		Partial Treatment	
	n	%	*n*	%
Palatal impingement	0	0	1	14
Gingival incisor contact	0	0	4	57
Dental incisor contact	38	88	2	29
No incisor contact	5	12	0	0

Figure 5 Case with partial treatment (OB > 50% at T2). Angle class II Division 1, skeletal class II, deep bite with gingival incisor contact, skeletal normodivergency, spacing in the upper and lower front, lack of space in the lower arch (left column). Initial treatment with upper plate with low pull headgear and lipbumper. After eruption of all permanent teeth the patient refused to continue with a multibracket appliance (middle column). 10 years later inflammation at the papilla incisiva is present due to palatal impingement (right column).

of malocclusion, mandibular growth rotation (Simons and Joondeph, 1973) and functional influences in combination

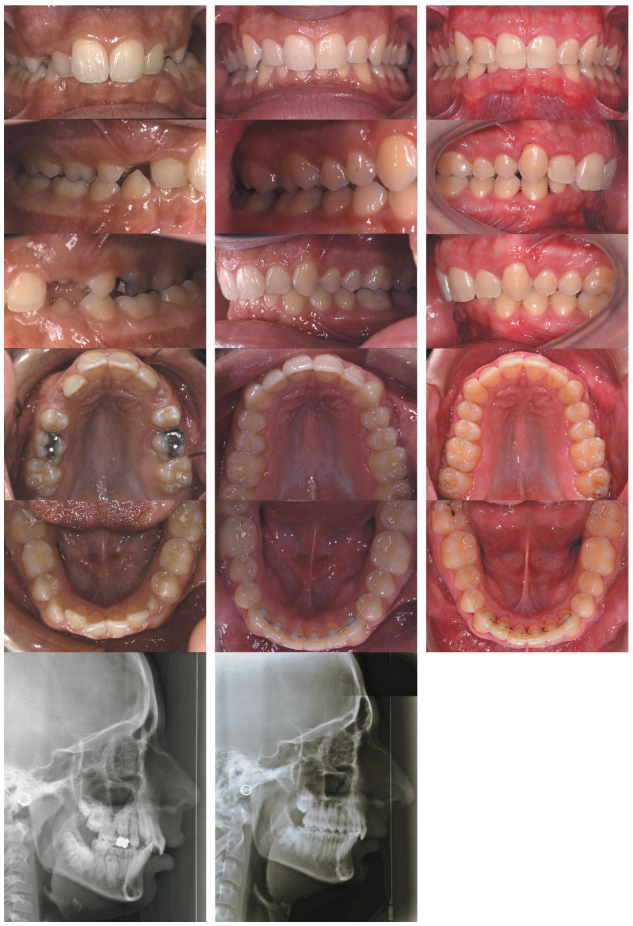

Figure 6 Twelve-year-old boy at T1 with partial treatment (OB > 50% at T2). Angle class II Division 2, skeletal class II, deep bite with gingival incisor contact, skeletal hypodivergency, unilateral crossbite, moderate frontal crowding in both arches (left column). The treatment was initiated by correcting the crossbite with a palatal arch and S-elastics. At the same time a low pull headgear was given to correct class II relationship, but success was poor due to missing compliance. Fixed appliances including base arch were used subsequently, but correction of class II relation could neither be achieved by class II elastics nor fixed class II mechanics. Active treatment had to be discontinued due to poor oral hygiene without full correction of deep bite and without establishing incisor contact at T2. At T2, deep bite was partially corrected. An upper bite plate for daily use and a functional appliance for night use was given as retention appliances. 13 years later incisors are in dentogingival contact and deep bite has returned to almost pre-treatment levels (long-term, right column).

with other malocclusions (e.g. lower lip pressure and sagittal bi- or unilateral relapse), and treatment outcome (e.g. type of incisor contact, interincisal angle, close interdigitation) or severe Bolton discrepancy may be clinically significant.

Relapse of the curve of Spee has been shown to be significantly increased in patients who were not completely leveled post-treatment (Preston *et al*., 2008). These findings may be similar to the comparison between the partial treatment group and complete treatment group in our study. It seems that patients who had partially corrected but persistent deep bite, e.g. due to treatment only in the upper jaw with persistent distal occlusion, or patients who

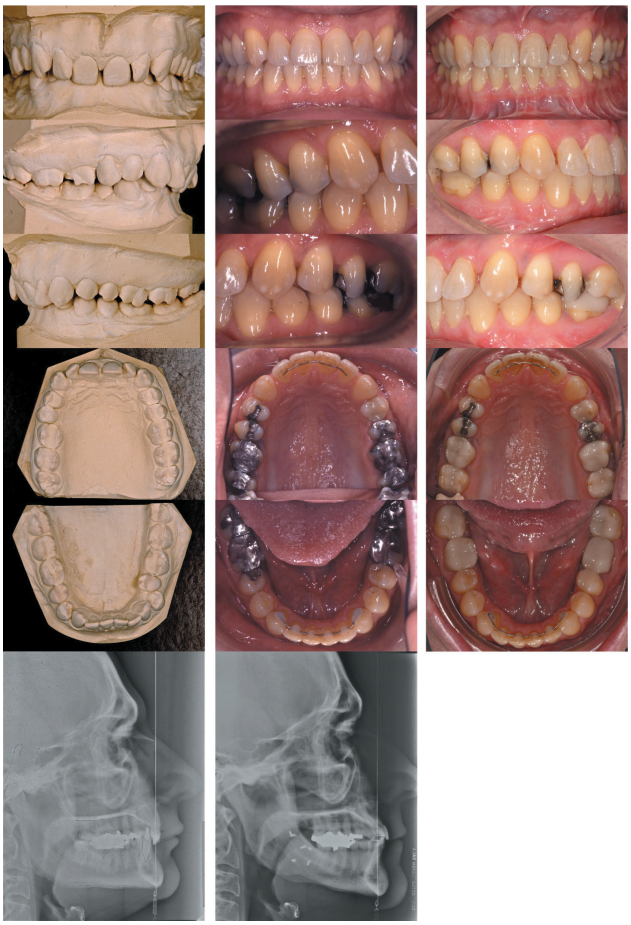

Figure 7 Adult case with long-term stability. Angle class II Division 2, skeletal class II, deep bite with palatal impingement, skeletal hypodivergency, maxillary and mandibular frontal crowding. Decompensation with multibracket appliance segmented arch technique, base arch and palatal power arms, extraction of third molars, surgery (sagittal split osteotomy and genioplasty), retention with upper and lower fixed retainers and retention plate (middle column). Excellent stability 11 years after treatment (right colum).

discontinued treatment or in whom treatment had to be stopped have a higher prevalence of gingival contact or palatal impingement at T3. On the other hand, it might be difficult to predict relapse, because spontaneous reduction in deep bite was noted in this study as well. An average decrease in overbite from 4.5 to 3.9 mm was reported in untreated class II Division 1 cases with deep bite between adolescence and adulthood (Feldmann *et al*., 1999), but this has to be interpreted with caution, because Class II Division 2 cases, which could be correlated with skeletal deep bite, were excluded. Similarly, Berg (Berg *et al*., 2008) reported a decrease in 9 out of 16 cases between the age 8 and 65 years. Although mandibular growth rotation (Björk, 1969) is difficult to assess without stable implants, the tendency for less relapse in high angle cases (Pollard *et al*., 2012) could indicate spontaneous bite opening by backward rotation during growth. It seems that higher variance of overbite in partially treated cases could be expected over time than in completely corrected cases. Observation and retention procedures to avoid relapse of deep bite in partially corrected cases seem therefore to be important unless backward rotating mandibular growth takes place.

It was not possible to identify factors of importance in the development of deep bite relapse due to the low prevalence and small amount of relapse. The stability rate of 90% in subjects successfully treated for deep bite may be partly related to selection bias during initial inclusion/exclusion or from informative loss to follow-up of 30% of patients. On the other hand, a bigger sample in this study may not change substantially the outcome because relapse was rare. In fact, a 6% higher definition of relapse in the setup protocol would have decreased the number in the relapse group to zero. Observer bias could not be fully avoided for timepoints T2 and T3 due to aging of the dental casts.

Conclusions

1. The prevalence of vertical relapse in moderate deep bite cases after a median post-treatment follow-up of 11.9 years was low (10.3%, relapse group $N = 4$).
2. The median deepening of incisor overlap in the relapse group at long-term follow-up (median 13.4 years) was low (6.7%, with a range from 3.2 to 19.8%).
3. It was not possible to identify important factors to predict relapse of deep bite malocclusion as prevalence and the amount of relapses were too low with respect to the sample, sample size, outcome, and retention procedures.
4. Deep bite at long-term follow-up was more likely due to partial correction at T2 (OB > 50% at T3, OB > 50% at T2, $N = 7$) than due to relapse (OB > 50% T3, OB < 50% at T2, $N = 4$).
5. Among all cases with deep bite at T3, gingival contact and palatal impingement were more prevalent in partially corrected noncompliant cases than in relapse cases.

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